Cataract development in Norwegian patients with congenital aniridia

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stability and inhibited retinal and choroidal neovascularization with similar efficacy as anti-VEGF antibodies (Rennel et al. 2011). Several studies have shown that the simultaneous blockade of Ang-2/Tie2 and VEGF pathways reduces vascular leakage and tumour sprouting angiogenesis and has an additive effect to single-agent treatment without apparent increased systemic toxicity (Koh et al. 2010). Combined inhibition of Tie-2 and VEGF signalling was also more effective in terminating pathological retinal neovascularization than anti-VEGF therapy alone (Takagi et al. 2003).

In this study, we performed vitrectomy for the management of sight-threatening complications of ischaemic RVO in four patients (Table 1). Vitreous haemorrhage, neovascularization and some fibrosis formation were observed in all RVO eyes. None of the patients had previous ophthalmologic history or diabetes prior to RVO. Intravitreal injections of anti-VEGF blockade, such as with bevacizumab or ranibizumab, were not used in any of the study eyes preoperatively. The intravitreal concentrations of both Ang-2 and VEGF were significantly higher in eyes undergoing primary vitrectomy due to complicated RVO compared with eyes with a quiescent idiopathic macular hole or pucker (p < 0.001; Table 1). This observation, together with the finding of normal intravitreal total protein and plasma Ang-2 levels among the patients with RVO, suggests that ischaemic retinal ECs secrete more Ang-2 than arises due to leakage from blood as a result of blood-retinal barrier breakdown. Interestingly, Ang-1, a factor with known vascular-protective properties, was induced in RVO eyes.

Based on these findings, we suggest that the combination of Ang-2 inhibitors with anti-VEGF therapy may be a more potent treatment for RVO than anti-VEGF therapy alone. However, clinical trials are needed to confirm our hypothesis.

Acknowledgements

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References


Cataract development in Norwegian patients with congenital aniridia

Ulla Edén,1 Neil Lagali,1 Anette Dellby,1 Tor P. Utne,2,3 Ruth Riise,4 Xiangiun Chen5 and Per Fagerholm1

1Department of Clinical and Experimental Medicine, Faculty of Health Sciences, Linköping University, Linköping, Sweden; 2Department of Medical Biochemistry, Oslo University Hospital, Oslo, Norway; 3Schepens Eye Research Institute, Massachusetts Eye and Ear Infirmary, Harvard Medical School, Boston, Massachusetts, USA; 4Department of Ophthalmology, Inland Hospital, Elverum, Norway; 5Syns laser Kirurgi AS, Oslo, Norway
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Findings in other patients included nuclear cataract (both turbid and yellow-brownish), one generalized subcapsular oedema (mature cataract) and one with a dehydrated opaque lens (hypermature cataract) (Fig. 1). In two patients, an anterior polar cataract was identified one of which had an additional posterior subcapsular opacification (Fig. 1). Of the 52 eyes examined, 25 had had surgical intervention (cataract, glaucoma or both). Nine patients (13 eyes) had cataract surgery only, six patients (eight eyes) both cataract and glaucoma surgery and three patients (four eyes) glaucoma surgery only. At the time of cataract surgery, 7 of 12 operated patients were under the age of 19 years and 4 of these were under the age of 10. Secondary cataract was observed in four patients (six eyes). Of the 25 eyes with surgical intervention, eight eyes (30%) had AAK affecting visual acuity compared to 8/25 eyes (32%) in the group of eyes without intraocular surgery. No clear trend could be found towards an increased prevalence of AAK in operated eyes.

Cataract is common in aniridia, with over 90% prevalence in our cohort, similar to a Korean cohort with 60 eyes where 88% had cataract or were operated for cataract (Park et al. 2010). Cataract prevalence in aniridia in the literature varies from 50 to 85% (Nelson et al. 1984).

Patients in our cohort not operated for cataract showed a distribution of lens opacities that could be interpreted as a pattern of cataract development. A discrete posterior polar opacity seems to emerge first. The posterior location of polar opacities has been reported previously (Yamn et al. 2011; Jin et al. 2012). The next phase is an additional subcapsular opacification in the mid-periphery. These opacities then increase in density and size, radiate to the polar region and are always limited to the posterior subcapsular region. They eventually form a ring on the posterior capsule.

References


Sale MM, Craig JE, Charlesworth JC et al. (2002): Broad phenotypic variability in a...
Severe retinopathy of prematurity in twin–twin transfusion syndrome after multiple blood transfusions

Arlette van Sorge,1 Frank Kerkhoff,2 Feico J Halbertsma3 and Nicoline Schalij-Delfos1

1Department of Ophthalmology, Leiden University Medical Center, Leiden, The Netherlands; 2Department of Ophthalmology, Maxima Medical Center, Veldhoven, The Netherlands; 3Department of Neonatology, Maxima Medical Center, Veldhoven, The Netherlands

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Table 1. Characteristics of the twins.

<table>
<thead>
<tr>
<th>Twin pair 1</th>
<th>Twin pair 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Donor</td>
<td>Recipient</td>
</tr>
<tr>
<td>Birth weight (g)</td>
<td>771</td>
</tr>
<tr>
<td>Gestational age (weeks)</td>
<td>28 + 1</td>
</tr>
<tr>
<td>Prenatal glucocorticoids</td>
<td>Yes</td>
</tr>
<tr>
<td>AV (days)</td>
<td>8</td>
</tr>
<tr>
<td>RDS (grade 2–3)</td>
<td>Yes</td>
</tr>
<tr>
<td>Inhaled NO</td>
<td>Yes</td>
</tr>
<tr>
<td>Transfusions</td>
<td>9</td>
</tr>
<tr>
<td>ROP</td>
<td></td>
</tr>
<tr>
<td>ROP stage</td>
<td>5</td>
</tr>
<tr>
<td>Plus disease</td>
<td>Yes</td>
</tr>
<tr>
<td>First screening</td>
<td>33 + 4</td>
</tr>
<tr>
<td>PMAROP</td>
<td>35 + 3</td>
</tr>
<tr>
<td>PMA severe ROP</td>
<td>41 + 5</td>
</tr>
<tr>
<td>ROP treatment</td>
<td>Laser</td>
</tr>
<tr>
<td>Lensectomy</td>
<td>Lensectomy</td>
</tr>
</tbody>
</table>

AV, artificial ventilation; ROP, retinopathy of prematurity; RDS, respiratory distress syndrome; PMA, postmenstrual age; Inhaled NO, inhaled nitric oxide; Severe ROP, ROP stage ≥ 3.